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INFECTIONS

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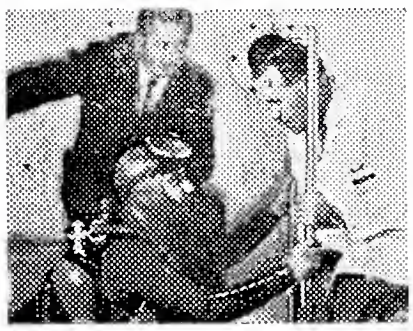

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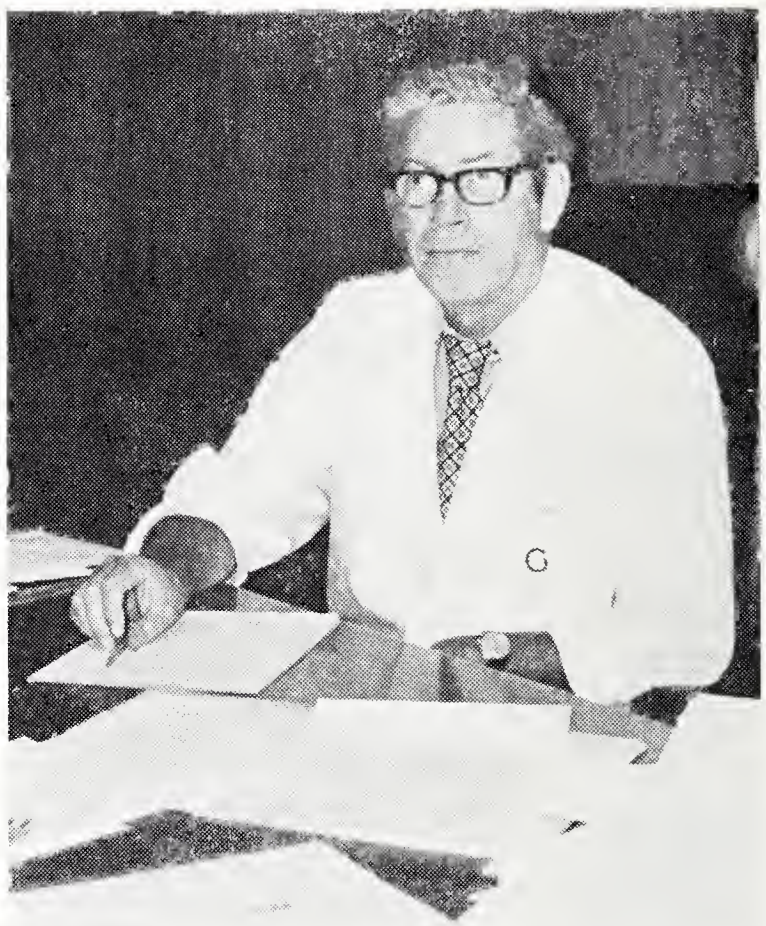
Guest Editorial

NORMAN A. HINTON,
M.D., C.M., M.Sc. (Med.)
Dip. Bact. (Lond.) F.R.C.P. (C)
Professor and Chariman,
Department of Medical Microbiology,
Faculty of Medicine, University of Toronto.
Microbiologist-in-Chief,
Toronto General Hospital.



Infectious Disease and Medical Microbiology — The Victory and the Challenge

No area of medicine or science demonstrates more clearly development in a dialectic pattern, than infectious disease. Dialectic proceeds, according to Hagel, by a necessary development, in stages, of thesis, antithesis and synthesis, which represents the process of developing thought moving toward completion. The history of medical practice represents a sequential series of responses to needs as they are are seen at the time, within the urgency of the immediate problem, utilizing and maximizing the *tools available at that time,* and dominated by the current "state of the art" as it reflects the stage of dialectic development.

Unless the above is clearly appreciated the present problem of the pattern of infectious disease represents a conundrum. Our recent history has been marked by a series of unprecedented successes in the prevention, diagnosis and treatment of a range of classes of infectious disease and yet the number of patients resident within our hospitals, suffering from serious infection, is not substantially reduced over what it was thirty years ago. Obviously the pattern has changed. The weight of the problem within the context of modern practice has not. This is the victory and the challenge.

At the turn of the century, infectious disease research and practice was heavily concentrated on the primary properties of microorganisms associated with their virulence and on the causation of disease through exposure of man to specific pathogenic agents. The induction of specific immunity in populations and the control of environmental contamination with particular pathogens evolved as part of this thesis. In the middle of the century potent antimicrobial therapeutic agents were developed and applied in the treatment of specific infections.

This line of development has been associated with substantial success. The immunization control of diptheria, pertussis, poliomyelitis, measles and smallpox; the environmental hygiene control of enteric fever, dysentery and cholera; the vector control of of plague, typhus and yellow fever; the reservoir control of domestic animal tuberculosis, brucellosis and rabies, and the dramatic antibiotic management of pneumococcal, streptococcal and other specific bacterial infections, has been associated with a major reduction in the morbidity and morality of primary exogenous infection. Classical postsurgical and postpartum deaths due to infection *in uncomplicated cases* have been reduced to statistically minute values.

The substantial successes recorded above, aside from the enormous benefit achieved, were associated with several significant misconceptions.

First, the reduction in classical epidemic exogenous disease was extrapolated by much of the profession to the whole of the area of infectious disease. The naive view became widespread that the thesis which resulted in the essential control of diphtheria, smallpox and typhus and the dramatic results of antibiotic treatment of pneumococcal and streptococcal disease need only be refined and extended. An important effort of this attitude was to progressively reduce the educational component in infectious disease in the medical schools, to discourage young physicians from entering postgraduate training in infectious disease and medical microbiology and to shift the emphasis in basic research away from primary study applicable in this field. The general expertise in the profession significantly declined. We are only now beginning to recover from this hiatus.

Second, the over-extension of the primary thesis to all levels of infectious disease created an entirely new range of problems, most clearly seen in the use of antimicrobials. Infection in man is common and the use of antibiotics to treat these infections involved the exposure of a large sector of the population to a new class of therapeutic agent. This exposure was further magnified by the massive application of antimicrobials as prophylactic agents and in the rote management of classes of patients (e.g., sore throats and fevers) with definitive diagnosis. Thus occurred an unprecedented exposure of the population to a whole new range of drugs whose toxicity and hypersensitivity reactions, and the complications of whose use, rose quickly to a primary place in iatrogenic disease.

Further, these biologically potent agents not only affected the specific pathogens against which they were used, but also acted as powerful selecting agents in the manipulation of the microbial flora of man and his environment, bringing to the fore antibiotic-resistant variants and species. This was probably the major factor in the genesis of the major pandemic of serious staphylococcal disease in the 1950's.

Many of the antibiotic-resistant organisms selected by antimicrobials and rising to dominance in the microbial flora of man during treatment or prophylaxis are not, however, in the classic sense, implicitly virulent. They may, however, populate an environment and may be effectively transmitted from person to person. Their significance rests in the antithesis to the view which dominated infectious disease at the turn of the century. Hippocrates held that infectious disease was primarily related to a disturbance of body function. This ancient belief was incorporated into a wholistic conception of infectious disease as the science evolved, postulating a balance between the virulence of the parasite, opportunity, mechanisms and circumstance of exposure, the number of organisms involved and the resistance of the host. As attention shifted from classical epidemic exogenous disease to the large background of endogenous, endemic, often hospital-acquired patterns, the importance of the latter component came to the fore. Urinary tract infection, wound infection, pulmonary infection in patients with pre-existing pulmonary and cardiovascular disease were increasingly recognized as a massive incidence of disease dominated by host susceptibility. Antimicrobial therapy in these cases obviously requires substantial discretion and judgment in order to obtain benefit without the result of fatal superinfection or cyclical infection with resistant organisms.

The problem of infection and host resistance has been further compounded by significant changes and advances in other areas of medical practice. The effective management of acute disease, whether it be infective, metabolic (i.e. diabetes) or nutritional, allows an older population to emerge, in which malignancy and cardiovascular disease predispose to important infective problems. The development of the corticosteroids and immunosuppressive agents, major advances in the chemotherapy of leukemia and other malignancies, major technical advances in intravascular, respiratory and cardiovascular support systems, the development of organ transplantation, important surgical advance in the implantation of artificial pacemakers, heart valves, hip joints — all carry with their benefit some significant increased risk of infection. Infection in the debilitated patient and often hospital-acquired disease, modified by antimicrobial therapy, is now one of the major problems of modern medicine.

The challenge of modern infectious disease and medical microbiology is formidable and requires more than ever a wholistic synthesis of the factors involved in infection. The progress achieved needs hardly to be emphasized. It is important to recognize that historical success does not define the objective of the profession or of medical care. We are committed to achieving whatever has been demonstrated to be achievable. Whatever is practically achievable *at the time* becomes the basic

standard of practice. *Problems* are defined as beyond this standard; *quality* is defined in terms of the degree to which one achieves beyond this standard.

The compendium of problems to be resolved is extensive. In terms of exogenous disease we are presently experiencing a massive pandemic of venereal disease. Common respiratory viral diseases remain essentially uncontrolled. Intestinal salmonellosis and food poisoning continue as major problems. The deep mycoses, Histoplasmosis, Blastomycosis and Actinomycosis, in a clinically significant form, occur at a low but essentially unchanged rate over the years.

Despite the knowledge of methods of control and treatment, major outbreaks of typhoid and cholera have been recorded during the past year. Indeed, in our own community, the fact that we do not yet have a system capable of "eliminating" an agent of infection, but possess a "control" system requiring major vigilance, is evidenced by the regular presence in our hospitals, albeit in small numbers, of cases of typhoid, pertussis and open tuberculosis. Diphtheria and tetanus are still recorded every year in Canada.

This problem is compounded by the increasing mobility of the world population so that a disease uncontrolled in any part of the world may be seen and require diagnosis and management in all parts. Cases of amoebiasis, filariasis, schistosomiasis, malaria, enteric fever, shigellosis and tuberculosis have all been seen in our clinics during the past year.

Many primary bacterial infections are now eminently treatable. This has not eliminated the *need* for effective diagnosis and treatment. Bacterial meningitis, streptococcal and pneumococcal infections are still common. Our ability to manage them effectively is at a high level of achievable capability and the required standard of performance in these infections, and in all others in which effective modes of treatment have been demonstrated, demands the very best laboratory and clinical performance.

Infection in the patient debilitated by pre-existing disease or the drugs, instrumentation, support systems or surgery required for his management is a major modern problem. In excess of 3 million hospitalized patients were estimated last year to fall into this category. Penicillin-resistant staphylococci continue to take their endemic toll. Gram-negative bacteria, involving growing numbers of patients infected with antibiotic-resistant Serratia, Klebsiella, Pseudomonas and Proteus, are seen in increasing numbers. Systemic disease in compromised patients may involve mortality rates of 30-60%.

Many other groups could be detailed. Surely the evidence is overwhelming that the challenge of infectious disease and medical microbiology is substantial. We must learn to wield the antibiotic weapon with discretion and skill. The gap between fundamental science and its application in the approach to the infected, debilitated patient must be closed. Major expertise, effective practice and important clinical and laboratory research must be developed and sustained.

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WEBSTER PRIZE

The editors are pleased to congratulate the winner of the Webster Prize for 1972-73, Dr. Paul Finnegan. The Webster Prize is awarded annually to the undergraduate author of the best contribution to THE JOURNAL.

We would also like to thank the Academy of Medicine for their continued interest and support.

S. W. EPSTEIN, M.D., F.R.C.P. (C)*

Lower Respiratory Tract Infections in the Intensive Care Unit

(NOTE: THIS IS A SUMMARY OF A PRESENTATION TO THE NORTH AMERICAN EASTERN CONFERENCE ON INFECTION CONTROL HELD IN TORONTO, ONTARIO, CANADA, OCTOBER 14 TO 16, 1973)

INTRODUCTION

Lower respiratory tract infections are a major problem in the intensive respiratory care unit. Their control and prevention is as important as their treatment in the management of this problem. The topic of control of respiratory tract infections in the intensive care unit will be covered here under the two major headings of 1. the problem and 2. our solution.

PROBLEM

The problem will be dealt with firstly from a general point of view and secondly by citing specific infections as examples.

GENERAL

There are two major reasons why respiratory tract infections are so important a challenge in the intensive respiratory care unit.

1. The first reason is poor host resistance. The patient seen in the intensive respiratory care unit often has multiple organ failure. The patient may have gone through recent trauma as a result of a car accident or surgery. In addition to an acute illness, there usually is a history of chronic debilitation due to alcoholism or some chronic underlying disease such as chronic airways obstruction, chronic congestive heart failure or leukemia. Often the lungs are the last organ to fail and when this occurs the patient transferred to the intensive respiratory care unit is in a state of severe body breakdown.

2. Secondly, there is an increased risk of infection. Many of these patients have an endotracheal tube in place or have had a tracheostomy performed. Thus, the upper natural barrier to infection is removed. In addition, there are many members of the health care team taking part in the care of the patient, each with their own potential risk of infection to the patient. Finally, there are also multiple forms of equipment used in patient care that can be viewed as fomites carrying infection to the patient.

SPECIFIC

CASE 1 – Staphylococcal Pneumonia

A 34 year-old female presented with a one-year history of increasing shortness of breath. Investigations six months previously showed an atrial septal defect and heart failure. Unfortunately, she had poor medical follow-up because of her own personal negligence. One week previous to her final admission she was visiting relatives in New York City. There she developed a fever, sputum and cough and shortness of breath. She went to a local hospital where they told her she should be admitted. However, she was afraid, as she did not have hospital insurance and took a bus back to Toronto.

She arrived in the emergency department in coma due to septic shock. Blood and sputum both grew *Staphylococcus pyogenes*. She was also in cardiac, respiratory and renal failure. She never regained consciousness despite intensive medical care including ventilation and appropriate antibiotics. She died seven days later of her Staphylococcal pneumonia and total body failure. Autopsy permission was not granted.

CASE 2 – Pseudomonas Pneumonia

A 42 year-old unemployed male gave an adult life-long history of poor nutrition and excessive alcoholic intake. In the previous six months he had been intoxicated constantly and had eaten very little. His mother, with whom he lived, brought him to the hospital because he had lost consciousness while vomiting bloody stomach contents when still intoxicated. He continued to vomit altered blood as well as to have melena. At the time of admission, his haemoglobin was 3 gms. %. He was resuscitated but continued to bleed and required surgery within eight hours for a vagotomy and pyloroplasty. His postoperative course was complicated by delirium tremens, as well as the previously noted aspiration pneumonia, respiratory failure and hepatic failure with coma. Despite intensive care including ventilatory support, he developed recurrent gastrointestinal bleeding from multiple stress ulcers and gradually deteriorated. Terminally, he developed an overwhelming Pseudomonas pneumonia which was confirmed at autopsy.

CASE 3 – Opportunistic Infections

An eighteen year-old boy who previously had had a renal transplant was on immunosuppressive therapy. He was admitted to hospital for investigation of fever and slight cough. Investigations noted diffuse pulmonary infiltrate on chest x-ray. An open lung biopsy

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*Assistant Professor, Department of Medicine, University of Toronto; Active Attending Staff, Department of Medicine, Toronto Western Hospital.

was performed and showed a *Pneumocystis carinii* infection. He was treated with Pentamidine isethionate and initially his chest x-ray remained stable and fever improved. Several days later the patient once again began to develop a fever. Repeated investigations including cultures were carried out. Increasing pulmonary infiltration was noted. He became semi-comatose, had a seizure, was intubated and despite intensive investigation and therapy, he died. Autopsy show widespread *Aspergillus fumigatus* infection as the terminal illness.

OUR SOLUTION

In the intensive respiratory care unit at the Toronto Western Hospital there are four beds in one room. There is a large open area with no physical confines between beds. This increases the risk of cross-infection. In addition to this four-bed room, there is available one room for isolation purposes. However, despite a very heavy case load of patients, often with infections that existed before they came into the unit, there has been very little difficulty recently with cross-infection from gram positive or gram negative bacteria. We still have occasional outbreaks of cross colonisation and the rare case of cross infection, thus indicating that we do not have the final, satisfactory solution. This good record has been maintained by following some of these procedures:

1. *Improved host resistance*

Probably the single most important feature in cutting down on infection is to improve the poor resistance of the host. Absolutely meticulous care of the patient is attempted through fluid and electrolyte balance, adequate nutrition, support of all body organs in order to maintain their functions within physiologically normal limits and early detection of infections.

2. *Careful use of antibiotics*

Phophylactic antibiotics are rarely used and antibiotics when used are usually based upon previous culture that is available or on the clinical grounds of the infection. Prolonged use of antibiotics is avoided.

3. *Reduction of formites*

Where possible, in order to avoid cross infection between patients, use of individual bedside equipment is preferred. This even includes providing the patient with his own stethoscope. However, probably the most important single item here is trying to provide one to one nursing. With the one nurse remaining with the one patient throughout the shift, rather than going back and forth between patients, contact and droplet spread are probably reduced.

4. *Careful suctioning technique*

It is important that the person who is going to do tracheal suction on the patient with a endotracheal or tracheostomy tube is well versed in the technique. This of course cuts down on trauma and improves on the sterility technique. The hands are washed and gloved and soft disposable catheters are used once. Masks are not worn.

5. *Isolation*

Patients with known Staphylococcal or Pseudomonas infection are removed from the unit as soon as possible. However, this is not always immediately practical because of the lack of isolation facilities. Certain patients who have very low resistance because of immunosuppression or burns are put in reverse isolation.

6. *Careful monitoring of sputum*

All patients in the unit have a culture of their tracheostomy site and sputum or bronchial aspirate twice a week. During their stay in the unit cultures from other sites as necessary are also performed. This includes initial examination for acidfast bacilli in all patients admitted to the unit.

7. *Sterilisation of ventilation and inhalation equipment*

This is done by the inhalation therapy department. The tubing on the ventilators are changed twice a day.

8. *Work with infection control team*

We are indeed fortunate to have an infection control team at the Toronto Western Hospital. Several studies have been carried out in the unit. As a result, Pseudomonas has been found in the hand cream, around the sink, on a fan, in a sheepskin mattress, in a glass containing false teeth, and of course, in cross contamination from other parts of the hospital, particularly the ventilators used in the operating rooms. These sources of infection were detected and eliminated.

Of course, in addition to all the above procedures, cleanliness of all personnel and of the rooms themselves is demanded. Visitors are kept to a minimum. Health care personnel try to wash their hands between examinations of patients and the rooms are washed and fogged as frequently as possible, particularly after any outbreak of infection. With these proceduces as noted above we have been able to maintain a fairly good record.

Congenital Toxoplasmosis

William C. Halliday, B.Sc., Dip. Bact. 7T5

BACKGROUND

In 1923 Dr. Josef Janku, a Czechoslavakian opthalmologist, described the first case of human toxoplasmosis. This infection was in the eye (chorioretinitis) of a congenitally infected infant. Dr. Wolf and associates, at Columbia University, described the first North American cases around 1939. A diagnosis of congenital toxoplasmosis was considered if hydrocephalus, chorioretinitis and intracranial calcification were present. These signs were referred to as the "classical triad". Over the years it has become apparent that the signs and symptoms of congenital toxoplasmosis vary greatly in a clinical spectrum from apparently normal to the severe neurological findings associated with the "classical triad".

THE MATERNAL INFECTION

How does the mother become infected? The answer to this question becomes apparent when one understands the life cycle of the coccidian *Toxoplasma gondii*. In birds, mammals and some reptiles there is an asexual cycle which contains the proliferative phase and the tissue cyst stage. The sexual cycle exists only in the intestinal epithelium of the feline host (e.g. the domestic cat). The sexual cycle contains schizogomy, gametogomy and the development of the oöcyst (see fig. 1).

Congenital toxoplasmosis occurs when the expectant mother develops acute toxoplasmosis during pregnancy (i.e. she has not been infected previously with *T. gondii*). This infection can come from eating uncooked or undercooked meat which contains infective tissue cycts. Alternately, it can be caused by ingesting infective oöcysts (i.e. oöcysts which have sporulated).

Unlike rubella, acute maternal toxoplasmosis in the first trimester of pregnancy is not likely to be transferred to the fetus. Danger of fetal infection arises when the mother becomes infected in the last half of pregnancy. The maternal infection rate for this infection is around one per cent. Maternal infection at this late stage carries a 40 to 50 per cent chance that the child will be born with either obvious or latent infection.

Studies to establish the incidence of congenital toxoplasmosis vary in their results. In Europe there have been studies showing incidence rates of between four and six per thousand live births. In the U.S.A., results indicate an incidence of one to two per thousand live births. Results can vary for climatic, ethnic, socioeconomic and technical reasons; the absolute numbers are not as important as the fact being presented — congenital toxoplasmosis can no longer be considered a "rare canary".

EFFECT OF TOXOPLASMOSIS ON THE PREGNANCY

What adverse effects does toxoplasmosis have on the pregnancy? This is a question that really cannot be given a definitive answer. With respect to stillbirths and abortions there are inconclusive and, in some instances, contradictory findings. Words such as "may," "appear," and "rarely" are commonly used in studies concerning these topics. However, it is established that prematurity occurs more frequently in mothers who have a high or rising toxoplasma antibody titre.

THE CONGENITAL INFECTION

When the infected baby is born it may show signs of obvious infection, however, if the infection is latent, it may appear normal. Of the babies infected during intrauterine life, about one-third will be obviously infected when born. Infection in normal appearing neonates can be detected by taking IgM levels in the umbilical cord serum. In the infected neonate the IgM is equal to or greater than 19.5 mgm. % and does not decline in serial neonatal samples.

The clinician presented with an apparently normal neonate has two possibilities before him. Either the baby has escaped infection or it has been infected, but the infection is latent. With respect to the latent infection, one can predict neither when it will manifest itself nor the severity and extent of symptomatology. IgM analysis allows the clinician to separate the non-infected infant from the infant with a latent infection.

Basically the sypmtomatology is divided into generalized and neurological symptoms. Each baby is an individual; he can show features of both types of symptoms or he can show predominantly features of one. The symptomatology can cover a spectrum from severe to very slight.

The symptoms classified as "generalized" are hepatosplenomegaly, lymphadenopathy, jaundice, anaemia, fever, vomiting and a masculopapular eruption. The visceral lesions are caused by cell necrosis resulting from intracellular trophozoite proliferation. As immunity develops there is the formation of tissue cysts and a state of premunition (infection-immunity) is achieved. The intact tissue cysts do not appear to provoke an inflammatory reaction. In the liver, a giant cell hepatitis with extramedullary haematopoiesis may be present.

The following ideas are presented as food for thought. *T. gondii* can produce extensive neurological destruction and thus cause a substantial neurological deficit. Certainly the neurological deficits will be noticed more readily than a slight anaemia or maculo-

papular rash. In cases with predominantly neurological findings, visceral organ involvement has been found at autopsy. The neural infection remains active longer in the brain and retina than in the other parts of the body as the 'benefit' of the development of immunity is delayed. Consequently the lesions may be more extensive and severe than in other organs. Observing these tragic neurological lesions, the conclusion one may make is that *T. gondii* has a predilection for neural tissue.

The neurological symptomatology includes chorioretinitis, hydrocephalus, intracerebral calcification — the classical triad — nystagmus, convulsions, lethargy and microcephalus. The neuropathology produced results from a) the proliferation of trophozoites and b) a Type III-like immunological reaction.

a) In response to intracellular proliferation resulting in cell necrosis, areas of necrotic cells are surrounded early by microglial cells and ultimately by astrocytes. Toxoplasma may be found in the periphery of these lesions. *T. gondii* infecting the intima of cerebral vessels may cause infarction of the cortex resulting from vessel thrombosis. The tissue cysts, once formed, do not elicit an inflammatory reaction from the host. However, a tissue cyst may rupture at any time and result in adjacent cell infection and necrosis which provokes an acute inflammatory reaction.

b) The type III reaction can give two different pictures depending upon whether or not there is cerebral aqueduct obstruction. These lesions are initiated by toxoplasmic encephalitis. The cerebral aqueduct can become obstructed secondary to periventricular and periaqueductal ulceration. The ependymal ulceration allows the toxoplasmic antigen to diffuse into the periaqueductal tissue and there react with a vascular factor (antibody). This causes tissue necrosis, vasculitis and thrombosis. Eventually the periaqueductal lesion leads to obstruction of the aqueduct.

The obstruction causes a non-communicating hydrocephalus to develop. The CSF rostal to this obstruction is generally xanthochromic and shows a pleocytosis of both red and predominantly mononuclear white blood cells. The protein is often in the range of one thousand mgm. %; however, it has been known to rise as high as two thousand mgm. %.

Within the obstructed ventricles there is a build up of the toxoplasmic antigenic substance. The antigen diffuses out into the periventricular tissue in areas of ependymal ulceration. Classically the lesions in this area are described as having four subzones. They are described as follows:

(i) Zone I is the farthest from the ventricular lumen and shows a cellular infiltrate.

(ii) Zone II shows vascular damage and consequent thrombosis.

(iii) Zone III is characterized by general cellular necrosis and possible calcium deposits.

(iv) Finally, zone IV, which is adjacent to the ventricular lumen, shows loose granulation tissue representative of an attempt at repair.

When aqueduct obstruction does not occur, the toxoplasmic antigen is allowed to disseminate throughout the CSF. The fourth ventricle may show periventricular lesions of the aforementioned type. Occasionally non-obstruction allows the development of microcephalus rather than hydrocephalus. The latter clinical picture may cause one to think of cytomegalovirus, which is more often associated with cerebral damage, non-obstruction and microcephalus.

The commonest symptom associated with congenital toxoplasmosis is chorioretinitis. It can be present at birth or it can develop as a late sequela. The damage to the eye may be localized or it may spread to involve the entire eye. It is estimated that one third to one half of the cases of chorioretinitis are due to toxoplasmosis. Chorioretinitis is found in about 86 per cent of newborns showing neurological symptoms. In the older patients it is difficult to establish if the chorioretinitis is the result of post-natal or congenital infection.

The final consideration is mental retardation caused by the congenital toxoplasmosis. Obviously, if the neurological damage is severe and the infant lives, he will be retarded. The real problem lies with those children who have symptomatically very mild infections and those children who are known to be infected, but have appeared normal since birth. The studies that have been done with these children suffer from the same inconclusive results as do those concerning abortion and stillbirth. An adequate study is difficult to achieve; however, it must be achieved before firm statements can be made concerning mental retardation and the subclinically infected child.

SUMMARY

1. Congenital toxoplasmosis is not as rare a disease as was once thought.

2. The maternal infection rate is about one per cent and 30 to 40 per cent of the maternal infections will cause fetal infection.

3. The mother can contract her infection from either ingesting infective tissue cysts (e.g. uncooked meat) or sporulated oöcysts (e.g. oral-faecal contamination).

4. The consequences of intrauterine infection are extremely variable. Desmonts, a French investigator, found that eight per cent die shortly after birth, six per cent live with sequelae, 27 per cent live without sequelae and 57 per cent escaped infection.

(Continued on page 13)

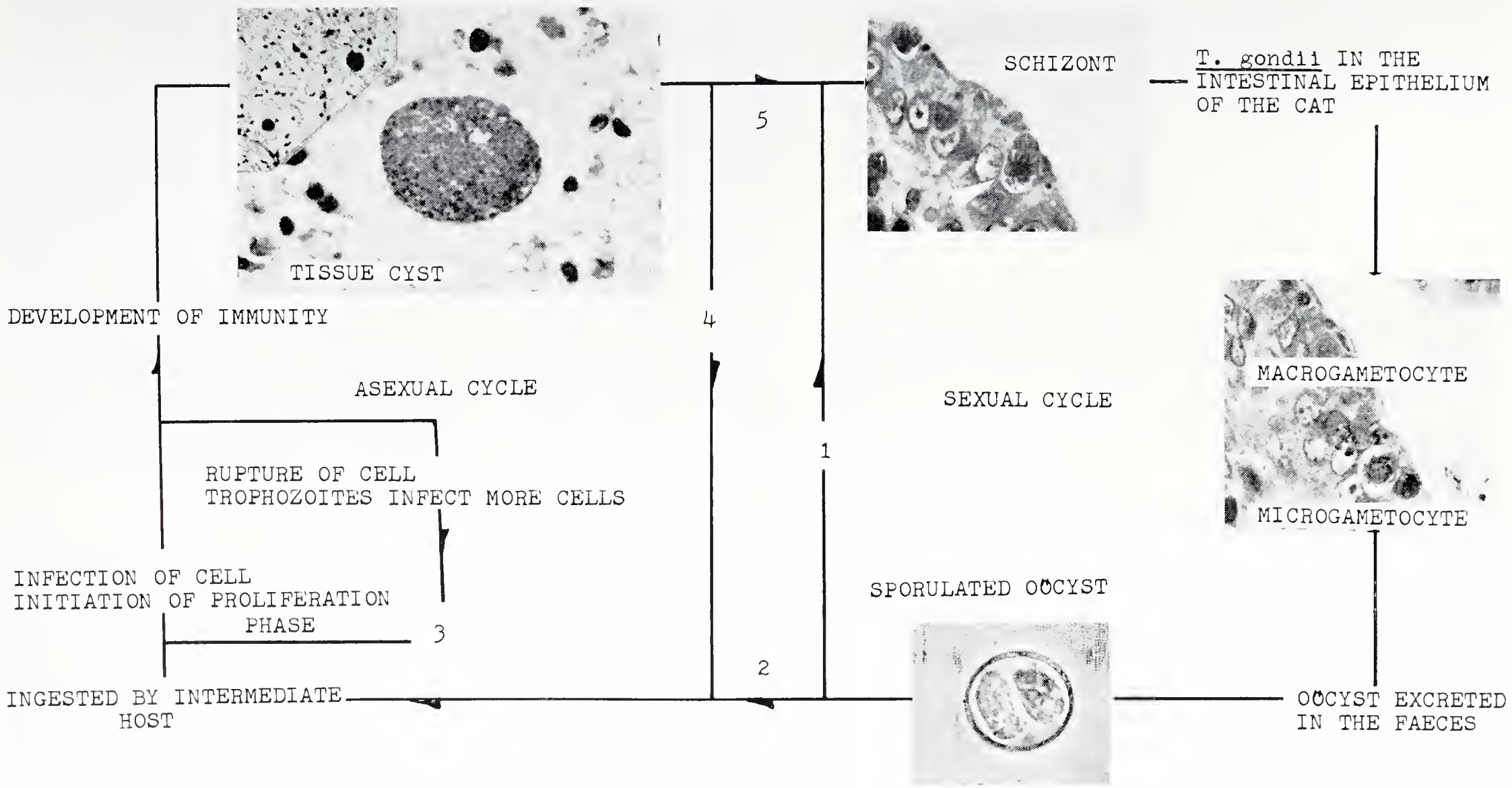


Fig. 1 Life Cycle of *Toxoplasma gondii*

1. If a cat ingests an infective oöcyst, then the sexual cycle is initiated.
2. If a non-feline host ingests an infective oöcyst, then an asexual cycle is initiated.
3. This is the proliferative phase and it is at this stage that a congenital infection can occur.
4. If a carnivore eats infected meat, then it becomes infected by the tissue cysts.
5. If the carnivore is a cat, then the sexual cycle is initiated.

Gonorrhea

*VIVIEN Y. H. CHANG, M.B.**
*K. F. GIVAN, M.D.**

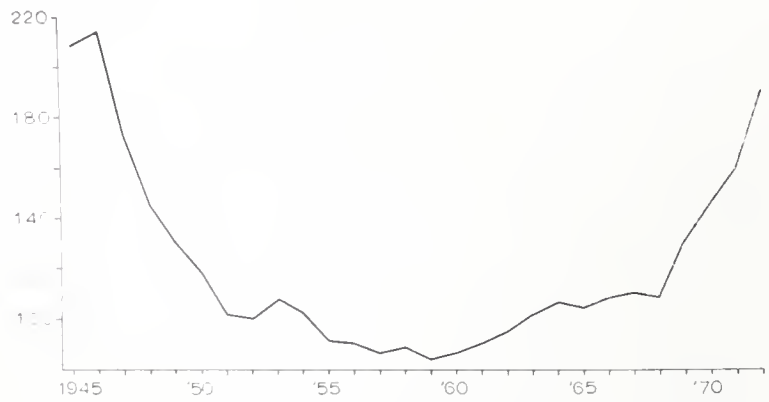
INTRODUCTION

Gonorrhea is the oldest venereal disease known to mankind. It was mentioned in the Bible and described by Hippocrates, but the word "Gonorrhea" (meaning a flow of semen) was first used by Galen. The term is a misnomer, but through prolonged usage it has defied any attempt to change it. This was also one of the first diseases to yield to chemotherapy, *Neisseria gonorrhoeae* being one of the most sensitive organisms to penicillin in the early days of introduction of this antibiotic. Naturally, one would then expect that eradication of the disease could be achieved without too much difficulty; on the contrary, a steady increase in incidence has been observed since 1957 and has reached pandemic proportions in the past few years. It is many times more common than syphilis and is now the most prevalent bacterial infection of adults as well as the number one reported communicable disease in North America and Europe, although we are well-acquainted with the causative agent and have effective methods of treatment. This article is written with the purpose of presenting the present difficulties and practical aspects as well as the possible new approaches that might be adopted for future control of this world-wide health problem.

INCIDENCE

Studies of the incidence rates of gonorrhea in Canada show a sharp decline between 1950 and 1957, followed by a gradual rise up to the present. The case rate per 100,000 population was 189.9 in 1972 (see Diagram 1). A parallelism applies also to Ontario; in 1972, 13,476 new cases were reported, corresponding to a case rate of 172 per 100,000 population — a tremendous increase over the reported case rate of 38 per 100,000 ten years ago (see Diagram 2). Alarming as these figures may appear, it is believed that they only account for 10% of the total number of cases because of gross under-reporting. It is estimated that approximately 150 million cases occur annually throughout the world.

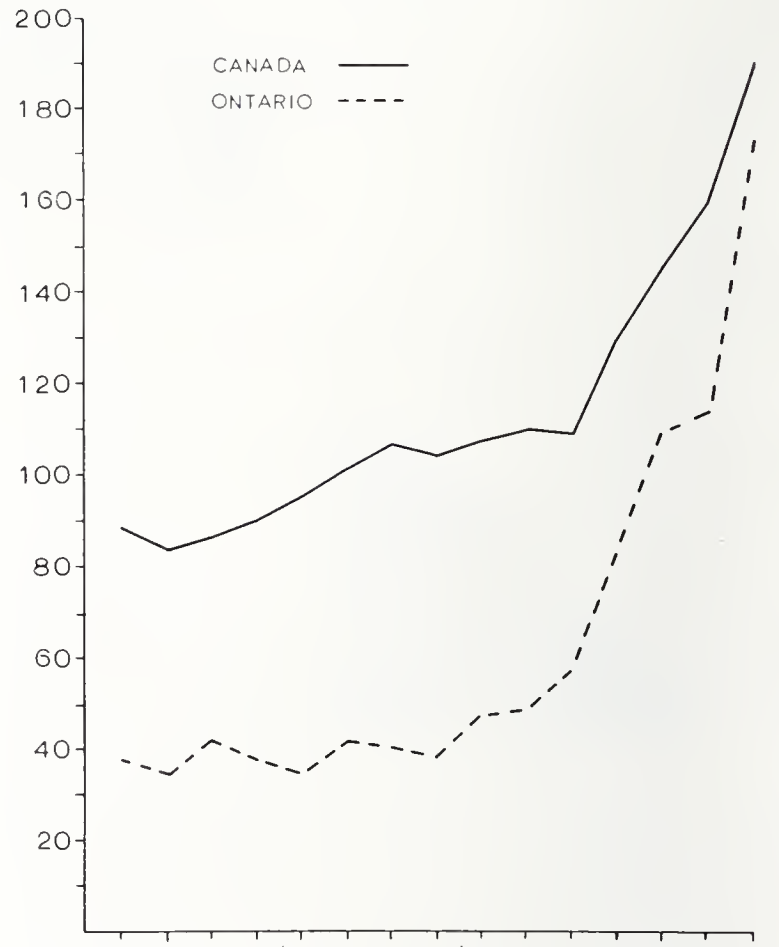
DIAGRAM 1.



GONORRHEA PER 100,000 POPULATION (CANADA)

Reported disease is concentrated in large cities with higher incidence in young adults of ages 20 to 24, followed by teenagers of 15 to 19 years. All segments of society are affected[1].

DIAGRAM 2.



GONORRHEA PER 100,000 POPULATION

EPIDEMIOLOGY

Gonorrhea is exclusively transmitted by sexual contact, except for conjunctivitis in the newborn infants and vulvovaginitis in pre-adolescent girls.

Man is the only known host. Asymptomatic human cases form the major reservoir of infection and are responsible for the main difficulty in eliminating this disease. Up to 90% of females infected with the organism are asymptomatic and it is now discovered that up to 10% of infected males are carriers, contrary to the former view that males always develop symptomatic infection if they contact the disease[2].

ETIOLOGY

The causative organism, *Neisserai gonorrhoeae*, is a gram-negative diplococcus, meaning 0.6 to 1 micron, nonmotile, noncapsulated and nonspore-forming. It has fastidious growth requirements and grows best aerobically under increased CO_2 tension at 35°C. Biochemically, the colonies are oxidase – and catalase – positive like all Neisseria species. The ability of gonococci to

**Division of Microbiology, Department of Pathology Women's College Hospital, 76 Granville, Toronto.*

ferment glucose only with the production of acid but not gas serves to distinguish it from the other members of the same genus.

CLINICAL PICTURE

The incubation period is usually from two to five days, but may be as soon as 1 day or as late as 2 weeks following sexual contact. In the male, acute gonorrhea presents with dysuria and purulent urethral discharge, dirty yellow in colour. Constitutional symptoms, if present, are mild. Local spread to prostate and epididymis results in prostatitis and epididymitis. Fibrosis following suppuration may produce stricture of the urethra. In the female, acute gonorrhea involves the urethra, Skene's glands, Bartholin's glands and the cervix, but the disease is modified by the anatomical site, local flora and other concomitant genital infection. Symptomatic urethritis occurs only in a small percentage of female patients. Cervicitis is the usual occurrence with mild to moderate amount of discharge. Occasionally local abscesses may form in the Skene's or Bartholin's glands. The vagina is not affected after puberty. Pelvic inflammatory disease may follow immediately after the acute infection or may be delayed for several months, often with involvement of both fallopian tubes, formation of pyosalpinx and pelvic peritonitis.

Proctitis, when present, may give rise to irritation and a purulent discharge and cannot be distinguished from inflammatory bowel disease such as ulcerative colitis when mucosal abnormality is confined to the rectum. Bacteriologic studies are necessary to establish the diagnosis under such circumstances. In a male, direct implantation by rectal intercourse is the obvious mode of transmission, whereas in women it may be due either to spread from the vaginal discharge or genitorectal exposures. Rectal cultures are positive in 20 to over 50% of women; in 6 to 10% they are the only indication of gonococcol infection[3].

Becoming more common nowadays is gonococcal pharyngitis resulting from oral-genital contact. In one study it was found in 22% of patients who practiced fellatio[2]. This is the reason why the patient's sexual habits should be determined and multiple sites need to be cultured.

Gonococcal infection, when confined locally to the urethra and cervix, is a self-limiting disease; it is only when gonococci spread to other sites of the body that they can cause serious medical problems. Dissemination can occur by the following routes:

(1) Direct transfer from infected mucosal surface to another as in the case of ophthalmia neonatorum where the organism is transferred from the cervix of the mother to the eyes of the infant during its passage through the birth canal.

(2) Local extension to the prostate and epididymis in the males, fallopian tubes, pelvic peritoneum, Bartholin's and Skene's glands in the female.

(3) Hematogenous spread to produce arthritis, endocarditis, meningitis or septicemia.

It appears that the advent of chemotherapy has caused a reduction in the frequency of gonococcal complications in relation to the number of cases of genital gonorrhea. The incidences of salpingitis, prostatitis and epididymitis have fallen from 15% in the pre-antibiotic era to 2 to 3% at present, and gonococcal arthritis now only occurs in 0.1 to 0.3%. However, the rise in the total number of cases of gonorrhea in the recent years accounts for an increase in the total number of cases of these gonococcal complications. (4)

DIAGNOSIS

The finding of typical intracellular gram-negative diplococci within the pus cells of the urethral discharge in a male presenting with acute urethritis is sufficient for making a presumptive diagnosis of gonorrhea, since there are no gram-negative diplococci normally present in the male genito-urinary tract. Although culture for the determination of antibiotic sensitivity is desirable in case standard treatment fails, chemotherapy can be started on the basis of a positive smear alone. If the patient has been on antibiotics recently, the smear is much less likely to be informative and culture is necessary for diagnosis.

In a female, smears are unreliable since saprophytic Neisseria and short gram-negative bacilli are indistinguishable from Neisseria gonorrhoeae morphologically. Cultures, preferably from multiple sites, are essential.

In order to obtain maximal yield from the cultures, the collection of appropriate specimens and correct method of handling them hereafter are of paramount importance. In the male, cultures should be obtained by gently scraping the anterior urethra with a sterile wire loop or cotton swab and streaking the exudate obtained on the surface of the selective antibiotic media. The rectum and pharynx should also be cultured if indicated by history and clinical features. In the female, the cervix is the single best site to culture and the rectum should also be cultured if possible. Since the commonly used lubricants may kill off the small number of organisms that are present, it is advisable to use water only for the insertion of the speculum. After removing any mucus or discharge from the cervical area, a cotton swab is inserted into the endocervical canal and moved about to ensure adequate sampling. The swab is then rolled across the culture plate in a large "Z" pattern. Studies show that a single cervical culture give 82% positive result; when multiple sites are cultured at a single visit the yield is increased to 90%. (5)

If direct inoculation onto the culture media is impracticable, the swab should be placed in a transport medium which will only maintain the viability of the organisms for a few hours. Unfortunately, no ideal transport method is as yet available.

The selective antibiotic medium specially designed for the primary isolation of gonococci was first developed by Thayer and Martin and represents a major advance in laboratory cultural techniques in handling this delicate organism. This is an enriched chocolate agar containing three antibiotics, vancomycin, colistin and nystatin, which suppress other contaminants and allow the growth of only the pathogenic species *Neisseria gonorrhoeae* and *meningitidis*. Trimethoprim may also be added to inhibit the spreading species of Proteus. Once inoculated, the culture plates must be immediately incubated at 35°C in 10% CO_2. The plates are examined after 48 hours incubation. Oxidase-positive colonies of gram-negative diplococci growing on the Thayer-Martin agar are almost diagnostic of gonococci if the specimen is from the genital area. Definitive identification is established by carbohydrate fermentation and specific fluorescent antibody staining. (6)

At present, no serological tests can be recommended in the diagnosis of gonorrhea. The complement-fixation test lacks specificity and sensitivity and may remain positive for years.

TREATMENT

Several principles must be strictly adhered to in the successful treatment of gonorrhea. The aim is to achieve adequate blood levels and tissue concentration for the necessary duration of activity by using a sufficiently large single dose of bactericidal antibiotic which can be administered to the patient under direct supervision. Giving suboptimal doses of an appropriate antibiotic or incorrect selection of antibiotics is courting the development of resistant strains. Tests of cure should be done on all treated patients to detect therapeutic failure.

Procaine penicillin still heads the list in the literature as the drug of choice for the treatment of all gonococcal infections despite the emergence of strains that are partially resistant. Prior to 1955 *in vitro* growth of most strains of gonococci were inhibited by 0.05 unit/ml. of penicillin. Since 1965 65% of isolates have shown resistance to 0.05 unit and 15% to 0.5 unit (7). The figures in Toronto quoted by Amies correspond closely to the experience in the other parts of the world. He found that in 1959 63% of isolates were sensitive to 0.01 unit, while in 1967 27% were resistant to 0.3 unit and 8% resistant to 1.0 (8). Studies have shown that these *in vitro* sensitivities of gonococci correlate well with the response of patients to treatment.

The advantages of penicillin lie in the low cost and low toxicity of the drug, as well as in its reported effectiveness in aborting incubating syphilis after administration of one intramuscular dose for the treatment of gonorrhea (9). However, the occurrence of hypersensitivity reactions together with the increasing dosage required to overcome the relatively resistant strains limit its usefulness.

Any of the following regimen may be used for acute uncomplicated gonococcal urethritis-cervicitis for both males and females (10):

1. 4.8 million units of aqueous penicillin G divided into 2 doses, given as intramuscular injections, one into each buttock at one visit together with 1 gram of probenecid given orally at least one half hour prior to the injection. (Note: oral penicillin and benzathine penicillin are not recommended.)
2. Oral ampicillin, 3.5 grams in one dose and 1 gram of probenecid administered simultaneously.
3. Spectinomycin dihydrochloride pentahydrate (Trobicin), 4 grams in one intramuscular injection.
4. Tetracycline hydrochloride, 1.5 grams stat followed by 0.5 grams q.6.h. until a total dose of 9 grams is administered. (Note: not recommended in pregnancy.)

In a pregnant woman, the safest drug to use is erythromycin (total recommended dose of 8 grams). Other antibiotics which may have to be considered include vibromycin, rovamycin and minocycline.

Test of cure should be made routinely three to seven days after completion of therapy by follow-up cultures from the sites where the organisms were isolated previously. Two negative cultures in males and three negative cultures in females are accepted as proof of cure (11). Contact tracing is an important part of the treatment in preventing re-infection and elimination of the reservoir of asymptomatic cases. It is the responsibility of the physician to report all cases of gonorrhea to the local health authorities.

The significance of ruling out co-existant syphilis cannot be over-emphasized. A serological test should always be done prior to therapy and then monthly follow-up for incubating syphilis for three consecutive months is advisable.

For complications of disseminated gonococcal infections, soluable penicillin should be administered parenterally in appropriate doses. Ophthalmia neonatorum caused by gonococci is an acute medical emergency and both parenteral and tropical therapy with aqueous penicillin G is indicated. The infected newborn should be isolated to prevent a nursery epidemic.

PREVENTION AND CONTROL

1. Adequate follow-up, diligent contact tracting and case finding are mandatory. Prophylactic treat-

ment to all contacts after appropriate diagnostic procedures have been carried out are proposed by some authorities. Routine screening in family planning clinics, cancer detection clinics and antenatal clinics are also suggested.

2. The development of a serological test for the detention of asymptomatic cases which may be used as a screening procedure in clinics together with VDRL is being investigated. The method has to be rapid, sensitive and as specific as possible so that large numbers of patients can be tested.

3. Mechanical prophylaxis with condoms appears to afford partial protection.

4. Prevention of ophthalmia neonatorum is achieved by routine instillation of 1% silver nitrate solution into the eyes of all newborn infants.

5. The development of an effective vaccine may well prove to be a major breakthrough in this area, but this is fraught with difficulties, as it is well-known that one attack of gonococcal infection does not confer any immunity against a repeated attack and antibodies are not often demonstrable.

6. Education of the public is conducive to earlier diagnosis of symptomatic disease and co-operation in contact tracing. More intensive teaching of venereology in medical schools is also urged.

7. It is hoped that in administering the appropriate antibiotics in doses effective against the most resistant strains the number of resistant isolates may be decreased. Since the limit of the amount of procaine penicillin that can be given at one clinic visit will very soon be exceeded, the reservation of a new antibiotic exclusively for the treatment of gonorrhea has been proposed.

CONCLUSION

Gonorrhea continues to present a major public health problem. A renewal appraisal in terms of epidemiology, as well as methods of prevention and effective control is urgently required.

ACKNOWLEDGEMENT

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(Continued from page 8)

5. A serially stable IgM of 19.5 mgm. % or greater is considered diagnostic of congenital infection.

6. The parents can be assured that they need not fear toxoplasmosis in subsequent pregnancies.

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"Tropical" Parasites in Arctic Man?

*REINO S. FREEMAN, M.A. Ph.D.**

Obviously there is something wrong either with the title, or with the concept that human parasites are all necessarily "tropical". Centainly due to air travel a physician almost anywhere on earth might see a patient with a disease contracted thousands of miles away, and in this sense tropical parasites might appear in the Arctic. This, however, is not the objective of this article. Rather it is to consider in general the factors associated with the distribution, spread and survival of protozoan and helminthic parasites of man and to relate these factors to the situation in the far north, illustrating why certain human parasites do occur there and speculating on what may happen in the future as the native people give up their old patterns of life and settle into more permanent settlements.

There is no disputing that severe human parasitoses are better known and probably more common in the warmer parts of the globe. Is this, however, because (1) conditions are inappropriate for parasite survival at higher latitudes, or (2) are there human parasites in the Arctic but for other reasons they are less pathogenic and hence go unnoticed, or (3) are other factors involved?

In order to understand the spread and maintenance of parasitic infections in any area, it is necessary to consider not only how parasites enter and leave the human body and what happens to them in the body that may lead to disease, but, and this is frequently overlooked, it is equally important to know what happens to the parasites when they are away from the human host. In order to do this adequately, one must know the life cycles of the parasites involved. There are ten basic protozoan and helminthic life cycle with important modifications, involving man as a host (Fig. 1); for more detail, one may consult any recent textbook[1]. The primary route of entry into man for many protoza and helminths (trematodes, cestodes and nematodes) is the mouth (cycles 1-5). Other protozoa, trematodes and nematodes enter through the skin cycles 6-8), one protozoan is transmitted venereally (cycle 9), and another transplacentally (cycle 10). Occasionally some parasites may enter inappropriate orifices, e.g., the pinworm entering the vagina. The primary point of exit of parasites from the human body is the anus (cycles 2 and 6; some protozoa, nematodes and most cestodes). Other parasites leave via the skin (cycles 3 and 8; protozoa and nematodes), the genital opening (cycles, 5, 7 and 9; a protozoan, trematode and nematode) and the mouth (cycle 1; protozoa, and rarely the lung fluke or other helminths. At least two nematodes, and several cestodes do not escape until after the infected human dies. Animal parasites differ from most microbes in that only a few species are immediately infective on passage from the body, i.e., transmitted by contact (cycles 1 and 2a; some protozoa and a few cestodes). Most parasites require either a period of maturation within the cyst or egg (cycle 2b; some protozoa and nematodes), or hatching with or without preceding development within the egg followed by growth either as a free-living organism (cycles 2b and 6a: some nematodes), or a period of development with or without increase in numbers in one or more non-human hosts (cycles 2, 3, 5-8; protozoa, trematodes ,cestodes, and nematodes). Obviously whether human parasites are successful or not in a given area, depends both on patterns of human behaviour which lead to successful transfer from host to host (be it human to human, human to animal, or animal to human), and also on conditions not directly associated with man, e.g., climate, soil, water and presence of other suitable hosts (vertebrate and invertebrate).

The biology of parasites in native people, especially in the Subarctic and Arctic extending from Alaska across Canada to Greenland, is poorly known, although surveys indicate that various protozoa and helminths are present[2-16] (Table I). What type(s) of life cycles do these parasites have? Might parasites with other types of life cycles be anticipated as a consequence of rapidly changing patterns of life among the native people?

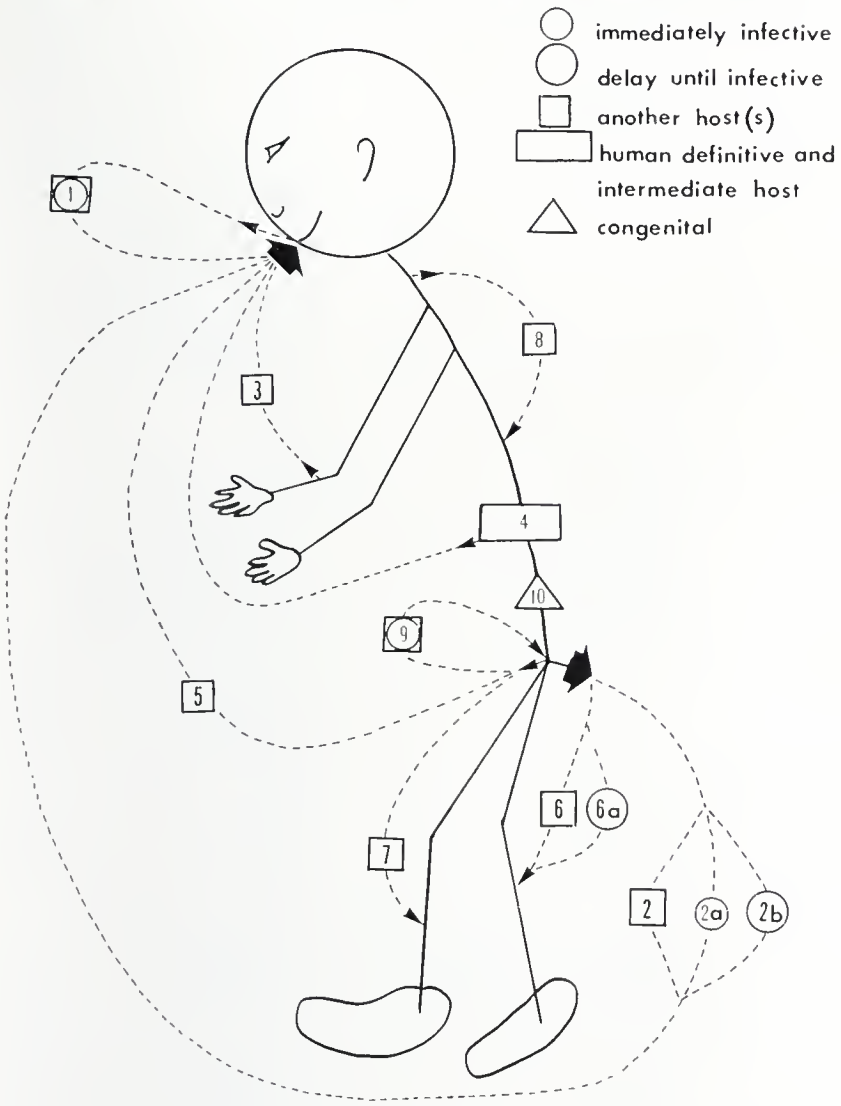
All the protozoa, except *Toxoplasma gondii*, (Table I) occur in the digestive tact, and all are immediately infective when passed in human feces, and re-enter the human body through the mouth (cycle 2a). However, all but *Dientamoeba fragilis* produce resistant cysts, indicating that they can survive for an extended time in water or a suitably moist environment before ingestion[1].

Toxoplasma gondii is relatively rare in the Subarctic and may be absent in the Arctic[17]. How it is transmitted there is not established, although in more temperate climates transmission to man occurs via ingesting either infected flesh, or oocysts passed in feline feces, or transplacentally from a recently infected mother to the fetus [17]. Presumably, there is a low incidence of infection among all animals at higher latitudes which propably accounts for the low incidence in man.

There are at least 43 species of trematodes which man acquires by eating improperly cooked fish, frogs crustacea[18] (Fig. 1, cycle 2), and two among them occur in arctic and subarctic North America (Table I). Other trematodes are acquired directly from water (Fig. 1, cycles 6 and 7) or by eating vegetation or

**Acting Chairman, Department of Parasitology School of Hygiene*

FIG. 1: REPRESENTATIVE EXAMPLES OF LIFE CYCLES IN MAJOR GROUPS OF PROTOZOA AND HELMINTHS INFECTING MAN



1.	MOUTH PROTOZOA –	ENTAMOEBA GINGIVALIS
2.	TREMATODE – LIVER FLUKE	METORCHIS CONJUNCTUS
	CESTODE – FISH TAPEWORM	DIPHYLLOBOTHRIUM LATUM
	NEMATODE –	GNATHOSTOMA SPINIGERUM
	PROTOZOA – AMOEBA	ENTAMOEBA HISTOLYTICA
	CESTODE – DWARF TAPEWORM	HYMENOLEPIS NANA
	NEMATODE – PINWORM (SLIGHT DELAY)	ENTEROBIUS VERMICULARIS
	PROTOZOA – COCCIDIAN	ISOSPORA HOMINIS
	NEMATODE – LARGE ROUND WORM	ASCARIS LUMBRICOIDES
3.	NEMATODE – GUINEA WORM	DRACUNCULUS MEDINENSIS
4.	CESTODE – HYDATID	ECHINOCOCCUS GRANULOSUS
	NEMATODE – TRICHINA WORM	TRICHINELLA SPIRALIS
5.	NEMATODE – KIDNEY WORM	DIOCTOPHYME RENALE
6.	TREMATODE – BLOOD FLUKE	SCHISTOSOMA MANSONI
	NEMATODE – HOOKWORM	ANCYLOSTOMA DUODENALE
7.	TREMATODE – BLOOD FLUKE	SCHISTOSOMA HAEMATOBIUM
8.	PROTOZOA – MALARIA	PLASMODIUM VIVAX
	NEMATODE – FILARIAL WORM	WUCHERERIA BANCROFTI
9.	PROTOZOA –	TRICHOMONAS VAGINALIS
10.	PROTOZOA –	TOXOPLASMA GONDII

snails, but these are unlikely sources of human infections under arctic conditions, as even further south in North America trematodes are uncommon human parasites. Snails are scarce or absent in the true Arctic, and since they are intermediate hosts for most trematodes, this probably accounts for the absence of flukes in such staple food fish as Arctic char in the vicinity of Igloolik, N.W.T.[6]

Quite unlike trematodes, cestodes, particularly various species of *Diphyllobothrium*, are common in arctic natives[2, 3, 4, 5, 6, 7, 8, 9, 12, 13, 15, 16] (Table I). All are acquired by eating uncooked or undercooked fish (Fig. 1, cycle 2). There has been confusion as to the specific identity of the species involved, it being assumed initially that most, if not all, were *Diphyllobothrium* (=*Dibothriocephalus*) *latum* which is well known from the Baltic countries in Europe. In fact, not many years ago it was assumed that this species probably was introduced to the Great Lakes area of North America by European immigrants. It is apparent, however, that there probably are species indigenous to North America[12] and that other circumpolar species occur in aboriginal North Americans, particularly in the Arctic. Proper identification of the species involved is more than a game played by biologists, as the various species differ considerably in their biology, e.g., how long they survive in the human gut. Undoubtedly, there will be isolated records of transient infections with other cestodes in arctic man acquired from animals that were eaten[12, 13]. Probably most human infections with *Diphyllobothrium* spp. are zoonoses, i.e., man being an incidental or accidental host in a life cycle maintained normally in native animals.

Human infections with *Echinoccus granulosus* and *E. multilocularsis* are an entirely different biological problem[19] (Table 1; Fig. 1, modified cycle 2 and 4). The former is better known and probably more common in arctic aborigines, but it may prove the less serious from the standpoint of human health[20]. Infections with both species are true zoonoses, in that the normal cycle for the former is between wolf and caribou, moose or other deer, and the latter cestode alternates between foxes and various rodents[21]. Eggs passed in carnivore feces are the source of human infection. It is unlikely, however, that human infection occurs directly from feces of wild carnivores, but rather from feces of dogs, which are synanthropic hosts[21, 22]. The latter becomes infected either by capturing and eating infected rodents, or from the common practice of eating the infected viscera of the herbivores killed by their masters. There is good reason to believe that

TABLE I
HUMAN PARASITES FROM NORTHERN CANADA AND ALASKA

Group	Species	Organ or System	References
Protozoa	*Entamoeba histolytica*	Digestive tract	5, 7, 10, 15
	Entamoeba hartmanni	,, ,,	10
	Entamoeba coli	,, ,,	3-10, 15
	Endolimax nana	,, ,,	3, 5-10, 15
	Iodamoeba büitschlii	,, ,,	7, 8, 15
	Dientamoeba fragilis	,, ,,	15
	Giardia lamblia	,, ,,	3-10, 15
	Chilomastix mesnili	,, ,,	9, 10
	Toxoplasma gondii	Various tissues	17
Trematoda	*Cryptocotyle lingua*	Intestine	13
	"Fluke eggs"	Intestine (?)	6
	Metorchis conjunctus	Liver	10, 18, 19
Cestoda	*Diphyllobothrium spp.*	Intestine	2-9, 13, 16, 19
	Diphyllobothrium alascense	,,	12, 16
	Diphyllobothrium cordatum	,,	16
	Diphyllobothrium dalliae	,,	12, 16
	Diphyllobothrium dendriticum	,,	9, 12
	Diphyllobothrium latum	,,	12
	Diphyllobothrium ursi	,,	12
	Schistocephalus solidus	,,	16
	Hymenolepis sp.	,,	16
	Echinococcus granulosus	Lung	8, 19-22
	Echinococcus multilocularis	Liver and elsewhere	19, 21
Nematoda	*Enterobius vermicularis*	Large intestine	3, 4, 5, 7, 8, 13, 19
	Anisakin larvae	Intestine	7, 13
	Trichocephalids (=Trichurids)	Intestine	13
	Trichinella spiralis	Musculature	7, 8, 11, 19, 21, 23

North American *E. granulosus* differs biologically from that found elsewhere[22], and it may be less pathogenic in man[20].

Two nematodes are often reported as parasites of North American subarctic and arctic aborigines[3, 4, 7, 8, 11, 19, 23] (Table I). Both have unique life cycles well adapted for cold climates. *Enterobius vermicularis*, the pinworm, has a 2a (slight delay) cycle (or alternatively a rapid 2b cycle) (Fig. 1). The eggs are unembryonated when laid by the female in a sticky substance, characteristically on the perianal region. This substance not only sticks the eggs to the skin, but also is an irritant which leads to scratching. Contaminated fingers are an effective mechanism for transferring the eggs to the mouth of the same or another individual[1, 19], particularly under the obligatory, intimate, housing conditions of family groups in the far north.

Trichinella spiralis is unusual in that it has no stage of development outside its hosts, transfer being direct from one meat eater to the next[11]. Although cycle 4 Fig. 1) shows that direct infection from man to man is possible, the opportunity for this is rare since most human cultures forbid the consumption of human flesh. Thus human infection in the north is a zoonosis resulting from consumption of infected flesh from wild animals, commonly bears[11, 19, 23]. Furthermore, since human flesh is rarely consumed by other animals, the normal cycle passes from one carnivore to another and continues to do so whether man is present or not. Rarely, other nematodes are reported from arctic man (Table I) but these probably are transient infections resulting from eating uncooked or undercooked fish[13].

Thus, human parasites with life cycles of type 2, 2a, 2b (modified) and 4 (Fig. 1) are known from subarctic and arctic North America. Quite likely, protozoa with cycles 1 and 9 are there also, but as yet are unreported. But why are parasites with other types of cycles absent? This is largely because of the short, frost-free period, unsuitable soil conditions for development of infective stages (cycles 2b, 6a), lack of snails, and the fact that in the high Arctic particularly, the water remains cold and the skin rarely is exposed directly to lake water (cycles 2, 6, 7). *Dracunculus* sp. and *Dioctophyme* sp. probably occur in certain aquatic animals in the subarctic (cycles 3, 5), but as yet they have not been reported from man in North America.

Human blood and tissue protozoa and filarial worms (cycle 8) have not been reported from the Arctic or Subarctic, although both types of parasites occur in other animals. Certainly anyone who has read about the north is aware that hordes of mosquitoes and other biting flies are present, and these transmit such parasites elsewhere. Conceivably the low density and widely scattered nature of the human population has precluded the evolution and establishment of such insect-transmitted parasites in man. The relatively few species of parasites which develop successfully in man in the Subarctic and Arctic are uniquely adapted and highly successful.

The culture and habits of the native peoples are changing and doing so most rapidly in the far north. Most people now live in relatively permanent villages, particularly during the winter, although family groups may spend short periods in hunting camps during the warmer months. Unfortunately, sewage facilities are relatively primitive, consisting usually of a "honey bucket" which is a plastic bag into which human excrement is collected and disposed of nearby — frequently on the sea ice. Water is obtained from a nearby lake, hauled as a liquid by vehicle during summer months, or as ice to be melted during winter. The central water tank in each house is not always very sanitary. The people have access to store supplies and usually have adequate fuel for heating and cooking. Apparently, this combination of factors has changed the pattern of parasitism, at least in the village of Igloolik, N.W.T. In 1949 the village was in the process of becoming permanent, and the people had a high incidence of fish tapeworm, pinworm, and head lice, but relatively few intestinal protozoa[3]. Some 20 years later, now that the village is well established with schools and a nursing station, the incidence of fish tapeworm, pinworms, and lice is low, but the incidence of intestinal protozoa is remarkably high[5, 6]. Furthermore, in Hall Beach, only 50 miles away, at least one infection with *Entamoeba histolytica,* a potentially severe pathogen, was diagnosed[5]. Incidentally, the latter village is adjacent to a major airport with people from other parts of Canada and U.S.A. moving in and out, providing ample opportunity for the introduction of parasites from further south. Less animal flesh is eaten uncooked today than 20 years ago, and the nursing station has brought medication to reduce the incidence of pinworms and lice. For people that remain largely on a hunting economy in the Arctic, the primary source of vitamin C is the raw fish or other flesh they eat[24]. Cooking destroys the parasites, and the vitamin C. A supplementary source of the vitamin will become necessary if the habit of eating raw flesh is given up entirely. The congregation of people and dogs into permanent, close-knit villages, the accumulating problem of sewage disposal and careless use of the water supply, also have introduced new problems, as has been shown. The changes in incidence and nature of the human parasite fauna are only one reflection of the changes taking place. The advantages of life in such settlements carries with it a price, and a changed and potentially dangerous burden of "new" parasites is only one of them.

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W. D. LEERS, M.D., Ph.D., F.R.C.P. (C), DIP. BACT., C.P.H.*

Salmonella Outbreak: A Case History

SUMMARY

In June 1968, an outbreak of salmonellosis due to *Salmonella typhimurium* occurred in the Wellesley and Princess Margaret Hospitals in Toronto, Ontario. There was a total of 146 cases in both hospitals. The Wellesley Hospital had 116 cases, 69 of them patients and 47 staff members. The Princess Margaret Hospital had a total of 30 cases comprised of 20 patients and 10 staff members.

The explosive outbreak was thought to be foodborne, originating from infected food and/or a human carrier from the dietary kitchen of the Wellesley Hospital. However, the source has never been found. *Salmonella typhimurium* was isolated from a reflux of a drain in the dietary area, that may have been responsible for the second wave of infection with salmonella. The outbreak was brought under control in a relatively short time by the initiation of vigorous hygienic measures and the isolation of infected persons. The total cost for the Wellesley Hospital alone was approximately $21,000, of which 40 per cent was due to workload in the microbiology laboratory.

I. INTRODUCTION

The prevalence of salmonellosis in man can only be estimated at the present time. In the U.S.A. there are an estimated two million human cases annually. Reported infections probably represent only a fraction of the total number, because the illness is often mild or even asymptomatic, usually self-limited and has a low mortality. Salmonellosis is rarely diagnosed clinically, except in epidemics. Bacteriological investigations are frequently not done, except where laboratory facilities are easily available.

At present, it is not known if all known salmonella serotypes are potential pathogens for man. *Salmonella typhimurium* has been found as the etiological agent more than any other serotype, and 12 serotypes account for 78 per cent of all documented cases of human salmonellosis[1].

During the period of 1963 to 1967, 40 epidemics of salmonellosis in hospitals and institutions, involving 3025 patients and 43 deaths, were reported from the United States Department of Health, Education and Welfare[2]. Epidemics occurred in newborn nurseries, pediatric and general hospital wards, nursing homes and mental institutions. Epidemics involving children were smaller and associated with a higher case-fatality ratio than outbreaks affecting adults. Contaminated eggs and egg products were responsible for most outbreaks occurring in adults. A total of 23 serotypes of salmonellae were implicated, with *Salmonella typhimurium* and *Salmonella derby* most frequently encountered.

The control of salmonellosis is difficult, because of the widespread distribution of the micro-organisms and the many diverse and indirect ways in which it can cause infection in animals and man. The reservoir of salmonellae in domestic and wild animals presents a major problem[3]. Control of salmonellosis is not achieved by interrupting animal to man transmission. Control of salmonellae in food or food handling would only reduce the incidence, but not eradicate the infection. Only education and high standards of hygiene would control person to person transmission[1].

There is no easy solution to the problem. Early detection and control of outbreaks require an efficient hospital infection control system. Contaminated food must be identified and removed, and human carriers must be found and isolated. Cross-infection outbreaks are difficult to control and require vigorous enforcement of hygienic measures and isolation of infected patients or suspects[4].

The epidemic of salmonellosis due to *Salmonella typhimurium* that occurred in the Wellesley and Princess Margaret Hospitals in Toronto in the summer of 1968 is described.

The "case history" is presented in a chronological order, followed by a discussion of the measures introduced and the difficulties that were encountered.

II. CASE HISTORY

1. *The outbreak:*

On the morning of Thursday, 6th June, 1968, salmonella was isolated from four stool specimens in the laboratory. The specimens were from in-patients from different wards of the Wellesley Hospital. In the early afternoon four more strains of salmonella were isolated from additional patients. Preliminary serological testing revealed that seven of the strains belonged to Group B and one to Group C. These strains were later identified as seven strains of *Salmonella typhimurium* and one *Salmonella bareilly*.

On the next day, five more strains were isolated, again from patients in different areas of the hospital. On the 9th June the first staff member was found to excrete salmonella. This staff member was a nursing assistant, who was found to have had gastro-intestinal symptoms for 2 to 3 days.

On the 11th June, the first salmonella was isolated from a patient of the Princess Margaret Hospital.

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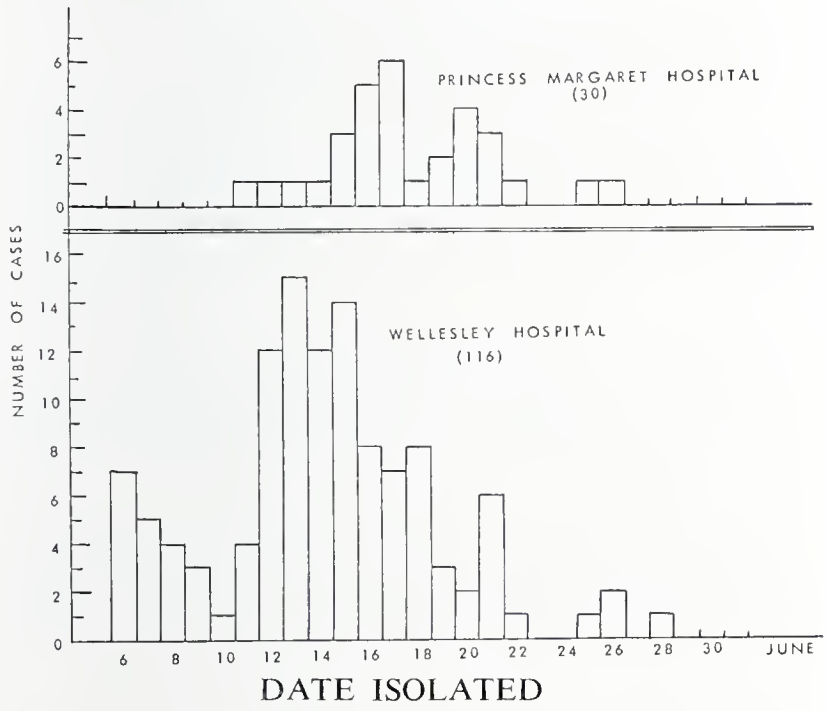
*Chief, Department of Microbiology, The Wellesley and Princess Margaret Hospitals, Toronto, Ontario; Assistant Professor, Department of Medical Microbiology Faculty of Medicine, University of Toronto, Toronto, Ontario.

On the 12th June, it was found that the assistant cook of the Wellesley Hospital kitchen excreted salmonella and, on direct interrogation, he admitted to having experienced diarrhoea, nausea and vomiting within the previous week. Several days later the cook was also found to be excreting salmonella. During this whole week, samples were taken repeatedly from kitchen areas, utensils and food in process, but all proved to be negative for salmonella.

Shortly after the onset of the epidemic, a backflow from the sewage drain was reported in the kitchen. This backflow was located in the cold storage room for vegetables. A swab taken from this drain contained *Salmonella typhimurium*. Investigation and discussion with the chief engineer revealed that several toilets were connected to this drainage system.

Approximately two weeks after the onset of the epidemic the number of stools or rectal swabs containing *Salmonella typhimurium* declined. The last positive specimen for salmonella from the Wellesley Hospital was found on 28th June and from the Princess Margaret Hospital on 26th June, 1968 (Fig. 1).

FIGURE 1
NUMBER OF S. TYPHIMURIUM ISOLATED FROM EACH HOSPITAL



2. *Measures taken to control the epidemic:*

a) ISOLATION OF PATIENTS

In the beginning, patients excreting salmonella were isolated on their respective wards. However, as soon as it became evident that an outbreak of salmonellosis was imminent, a special isolation ward was initiated. All patients with typical gastro-intestinal symptoms were transmitted to this ward until it was proven by the laboratory that they did *not* excrete salmonella. The typical clinical symptoms included diarrhoea, nausea, vomiting and abdominal cramps.

At the peak of the epidemic, three wards had to be used as isolation wards with a total of 100 beds. Disposable trays and gowns were used and isolation procedures were instituted and strictly enforced. Visiting was restricted to close relatives only. Everyone had to wear a gown and had to clean his hands on entering and leaving the room, under the supervision of a member of the nursing staff. The whole situation was very confusing for many visitors and, surely, for many staff members.

b) SEARCH FOR SOURCE (FOOD OR CARRIER)

Samples were taken from many areas of the kitchen and from the gastric diet and its separate ingredients. All were negative for salmonella.

c) STAFF CONTROL

Stool specimens were requested only from those of the medical and nursing staff who were in close contact with patients with salmonellosis. On the other hand, *all* dietary and *all* housekeeping staff were requested to send their stool specimens. In addition, all staff members with clinical symptoms suggestive of salmonella infection were sent home immediately and followed by the City Quarantine Officer and their own attending physicians.

Soon it became evident that the stool specimens requested, especially those from the dietary and housekeeping staff, were *not* being submitted. Therefore, a team of two technologists was set up to obtain rectal swabs on the wards and from staff. The rectal swabs were also preferable to the stool specimens, since a large number of stool containers were collecting in the laboratory, and since it was mid-summer, the aroma in the air was not the most pleasant. We were aware that rectal swabs may yield a lower number of positives if the number of salmonellae excreted is relatively low, as is often found in carriers[5]. It could be assumed, however, that during the epidemic, the number of salmonellae excreted was high enough to yield positive results by this method.

d) LABORATORY PROCEDURES

It was obvious within the first days of the epidemic that adjustments had to be made to cope with the work load, without jeopardizing the routine work of the laboratory. Therefore, the charge technologist was made responsible for the technical aspects of the salmonella problem and the chief technologist for the efficiency of the routine laboratory.

e) ADMISSION POLICY

Admission policies were reviewed in view of the epidemic. It was decided that it was unnecessary to restrict admissions to the hospital.

f) OTHER MEASURES

Numerous meetings were held with the Senior

Medical Staff and that of Nursing, Administration, Housekeeping and others during the epidemic. These were very time-consuming, but necessary to get the epidemic under control. The Infection Control Committee with the microbiologist as chairman was the leading group for instituting measures and regulations.

g) OUTSIDE CONTACTS

As soon as the eight cases of salmonellosis were found, the Medical Officer of Health at City Hall was contacted. It was important that the Public Health Authorities take the responsibility of looking after those patients that were discharged from the hospital and the staff members who were sent home and treated by their own attending physician. In addition, there may have been outside cases of salmonella infection in families of staff members or discharged patients.

When it became evident that an epidemic was imminent, we contacted the Ontario Department of Epidemiology and outlined our problem. Dr. Bell, at that time Provincial Epidemiologist, and his staff attacked the problem from the epidemiological aspect by investigating the onset of the symptoms of the patient (Table 2).

h) FOLLOW-UP OF STAFF AND PATIENTS

A discharge or re-employment policy had to be established. For persons who had not been treated with antibiotics, two consecutive stool samples or rectal swabs, at least 24 hours apart, had to be negative for salmonella before the person was considered not to be a carrier. Persons on antibiotics had to wait five days after cessation of therapy before the stool samples could be accepted for bacteriological examination.

Patients infected with salmonellosis were sent home, if they wished, but, as previously indicated, were followed by their own physicians and Public Health Authorities. Staff members sent home were not allowed to go back to work unless they had two consecutive stool specimens which were free of salmonella. The staff specimens were examined in our own hospital laboratory. In this way, we had a firm control over any staff member before he or she was allowed to return to work in the hospital. On some occasions, excretion of salmonella was found up to three months after the first isolation. However, no estimate as to the number of patients involved or the correct duration of the carrier state could be made, since many of the patients were followed up outside the hospital.

III. DISCUSSION

1. *The Hospital*

In order to understand some of our problems, some background information is necessary. The Wellesley Hospital and the Princess Margaret Hospital are two hospitals with two different administrations, located in one building complex. There are many shared services, and flow of staff from one hospital to the other is common. Also some food for the Princess Margaret Hospital is prepared in the dietary kitchen of the Wellesley Hospital.

At the time of the outbreak, the Princess Margaret Hospital had a stable 120 beds, and most of the staff had had many years of service in this hospital. Therefore communication presented few problems.

On the other hand, the Wellesley Hospital was, until 1966, a small hospital with 303 beds, which was enlarged to 602 beds in 1968. This increase in size demanded an increase in new staff and presented many problems for the administrative and senior staff who suddenly had to cope with many problems associated with a large hospital.

As soon as the first case of salmonella was found at the Princess Margaret Hospital, the Princess Margaret Hospital was sealed off from the Wellesley Hospital. The measures taken at both hospitals were much the same.

2. *Hospital kitchen*

There were many areas in the kitchen that could have been the source of the outbreak; for example, the meat in the refrigerator was stored in containers on the floor. The vegetables in the vegetable cold room were kept in baskets that were wet from water on the floor, water which may have come from the backflow of the drainage in the cold room. We learned later that there were repeated problems with reflux through several outlets in the kitchen in the months before the outbreak of the epidemic. It was in the vegetable cold room that *Salmonella typhimurium* was isolated from the reflux of the drain in the first week of the epidemic. The washrooms in the dietary area were found to be improperly cleaned. Food consumption in those "rest rooms" by members of the dietary staff was also a good possibility.

However, corrective measures were implemented as soon as possible and all areas in the Dietary Department were brought into a satisfactory condition.

3. *Staff and Employee Health Service*

The Employee Health Service presented a problem in itself. At that time there was no full-time physician in charge of this unit. The head nurse in charge had been in this position for only four months. Therefore, the microbiologist had the additional task of heading and directing the Employee Health Service during the epidemic. This was not an easy undertaking.

Another problem with dietary and housekeeping staff was that these people did not want to be sent home because they feared they would lose wages. Therefore, symptoms frequently were not reported. This was one of the reasons that it was decided to have all the

dietary and housekeeping staff (and not only those who reported symptoms) provide a faecal specimen.

Staff members who were sent home because of salmonella infection were paid through Workmen's Compensation. The hospital paid the difference between their regular salary and the amount paid by Workmen's Compensation.

4. *Education of staff and patients*

All transfers of patients were not only planned by senior nursing service, but personally supervised. Careful instructions were given to those transferring the infected patients. At least one graduate nurse accompanied every patient. The Director of Inservice Education found that a great deal of her time was spent reassuring patients who were being transferred. In addition to the staff education on the isolation ward, a programme of handwashing technique was introduced, which included discussion on faecal-oral contamination.

5. *Laboratory Problems*

The increasing number of specimens, work load, telephone enquiries and personal visits by staff forced us to restrict admittance of persons to the laboratory. Phone reports pertaining to the epidemic only were reported to the floor twice per day.

At the end of each day, a full written report of all positives was given to the Nursing Office.

Apart from the tremendous increase in laboratory work, there was also a great increase in clerical work, that was handled with a minimum of confusion considering the number of lists and reports that had to be made.

At all times, the hospital administration co-operated and offered help on request, but the introduction of new personnel had its obvious limitations. Help was also offered and accepted from the Technologist Training School, which provided four experienced senior technologists under the direction of Mrs. Harriet Smith, at that time Director of the Microbiology Section.

6. *Public Relations*

At a meeting with the Microbiologist, the Associate Administrator and Head of Nursing, it was decided to release *only* authorized information to relatives and to the public. The Director of Public Relations was authorized as the only person in the hospital to give out official information at a specified hour every day, after consulting with the respective staff members.

But very soon after the first detection of salmonella in the hospital, the story was in the newspapers, most of which reported that there was a "food poisoning" outbreak at the Wellesley Hospital. Information could have leaked out in several ways:

(i) We reported the first eight cases to the office of the Medical Officer of Health at City Hall on the first day, Thursday, June 6, 1968. It is conceivable that the information was obtained from there.

(ii) There was a rumour that a newspaper reporter was a patient in the Wellesley Hospital at the time of the outbreak. If true, this could have been the leak.

(iii) Relatives of either employees or patients could have talked to the press.

Unfortunately, with some exceptions, the communications media developed a habit of referring to the outbreak as due to "food poisoning" although nobody had any idea what the cause was. Another matter that caused us some misgivings, was the newspapers' habit of adding new cases of salmonellosis to old cases and coming up with a larger figure which included all the Wellesley patients and staff members who had contracted the infection, even though they had been sent home. This gave an impression that the infection in the hospital was far worse than it really was. As time went on, corrections were made and matters improved. In spite of all our difficulties, we were able to keep a cool head, since the Director of Public Relations handled the job extremely well, in spite of much frustration.

7. *Measures and policies instituted after the outbreak*

The most urgent steps were to correct the deficiencies and unsatisfactory sanitary conditions that could play a role in the spread of infection. The most important measures were to install racks in the cold rooms to remove the vegetables and meat from the floor and to provide the additional handwashing facilities in the washrooms of the dietary area. The rules and regulations of the housekeeping staff were revised and improved. In addition, a full-time physician was appointed to head the Employee Health Service, and many other improvements were instituted.

After the outbreak, the Administrator of the Wellesley Hospital called a meeting involving the chiefs of staff, senior administrators, the Microbiologist, the Chairman of the Infection Control Committee, and the senior nursing staff to establish a policy for the control of an epidemic.

8. *Expenses*

The epidemic was a great expense to both hospitals. It cost the Wellesley Hospital about 21,000 dollars, and laboratory expenses represented about 40 per cent of this total. The monthly work load in the microbiology laboratory increased from approximately 3,000 specimens in May 1968 to approximately 4,000 specimens in June and July. Most of these additional specimens were stool samples or rectal swabs.

10. *Source of outbreak*

The source of the salmonella epidemic in the Wellesley Hospital and Princess Margaret Hospital remains speculative.

The fact that several cases occurred explosively in the hospital on unrelated wards made food from the kitchen a distinct possibility. The only common food vehicle consumed by some patients was a special gastric diet made up of basic foods including eggs, milk, fruit, strained vegetables, fish and meat, prepared in our own dietary kitchen. A culture of the ingredients revealed no salmonella, but no part of the original gastric diet could be retrieved for bacteriological examination.

The first positive rectal swabs or stool specimens with salmonella from the staff came exclusively from the dietary staff members. There were 19 staff members in the first six days of the epidemic who excreted salmonella, 15 of them from the dietary staff. The cook and assistant cook were excreting salmonella also, the latter having had symptoms that were suggestive of gastroenteritis several days before he submitted his stool specimen.

TABLE I
DISTRIBUTION OF SALMONELLA INFECTION AMONG STAFF OF THE WELLESLEY HOSPITAL AND PRINCESS MARGARET HOSPITAL 1968

Classification	Total Number Wellesley Hospital	Princess Margaret Hospital	Number with clinical symptoms
Dietary	17	1	3
Nursing and Hospital Assistants	18	2	11
Housekeeping	5	2	0
Physician	1	0	0
Other	6	5	2
	47	10	16

Table I shows the distribution of salmonella infection among staff of the Wellesley and Princess Margaret Hospitals during the epidemic.

One could speculate that there was a human carrier in the dietary kitchen who was preparing the food. Who this person was and if he or she were acutely ill or an asymptomatic carrier, is open to speculation. But, contamination of food by a human carrier at the time and place of preparation is a distinct possibility. Improperly processed meat or poultry containing salmonella, imported into the kitchen from outside, is another possibility.

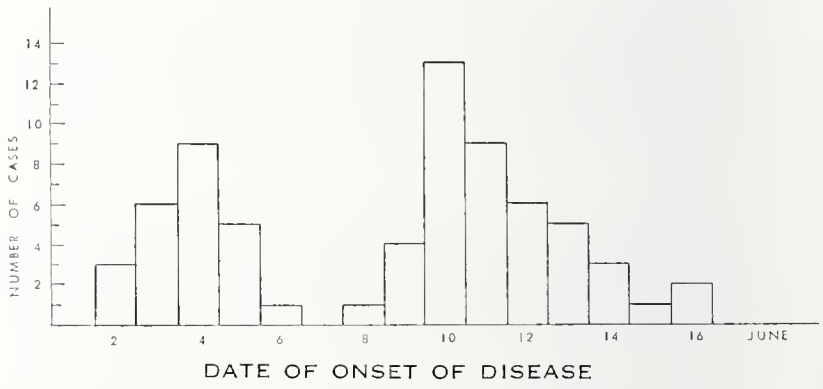
The infection in the Princess Margaret Hospital probably was started by contaminated food that was delivered from the Wellesley Hospital kitchen. The delivery of food was stopped as soon as the first case was found at the Princess Margaret Hospital. This resulted in relatively few cases at the Princess Margaret Hospital and a short outbreak only.

Why did the dietary staff have the highest percentage of salmonella? This was probably due to the cross-infection due to poor hygiene, improper handwashing and contaminated toilets in the dietary area.

As was indicated before, we found conditions in the washrooms of the dietary area unclean, thus making this assumption highly probable. The cross-infection in the dietary staff probably gave rise to additional sporadic infections on the wards due to contamination of additional food. The other hospital staff members probably became infected through contaminated food consumed in the Wellesley Hospital cafeteria.

The fact that we had two peaks in the graph during the epidemic indicates a second wave of infection after the first one subsided (Figure 2). The *first* wave was probably due to contaminated food, possibly to one human carrier. Infected eggs, meat or poultry improperly processed before or during preparation is another remote possibility.

FIGURE 2
ON SET OF CLINICAL DISEASE BY DAY FOR 68 CASES OF SALMONELLOSIS
(Wellesley Hospital, 57 Patients – Princess Margaret Hospital, 11 Patients)



The second phase could have started in several ways. The most likely are:

- The cross-infected dietary staff contaminated additional food during handling.
- The backflow of the drain that was found to contain salmonella contaminated the vegetables and/or meat in the cold room of the kitchen and from there contaminated food reached the patient.

11. *Conclusion*

Fortunately the epidemic was controlled within three weeks by sending every suspect home and by enforcing good sanitary conditions wherever possible. As can be imagined, many of those hygienic measures to be implemented took time, patience and education. Such an epidemic may happen to any hospital or institution.

(Continued on page 24)

KATHLEEN F. GIVAN, M.D., F.R.C.P.(C)*

Streptococcus Group B — A New Threat to the Newborn

In the past two years Streptococcus group B has emerged as an important cause of neonatal sepsis. This article reviews the recent evidence incriminating this organism.

BACKGROUND

Prior to the use of antibiotics, Streptococcus group A was the single most important organism responsible for neonatal sepsis. With the introduction of penicillin, Group A streptococcus became rare as a cause of neonatal infection. In its place, Gram negative enteric organisms, especially *E. coli* became the most important agents. The pattern changed once again during the 1950's when due to indiscriminate use of antibiotics a world-wide epidemic of Staphylococcus aureus occurred, affecting many major hospitals.

These strains of Staphylococcus, especially strain 80/81, were resistant to most of the antibiotics then in use. The introduction of penicillinase-resistant penicillins, the more rational use of antibiotics, and perhaps changes in the virulence of the organisms themselves brought about a decline in the incidence of Staphylococcal infections, and once again the Gram negative organisms become the prevalent ones in nursery infections. However, in recent months a number of centres have reported Streptococcus group B to be the single most important organisms in the etiology of neonatal sepisis.

BACTERIOLOGY

The Streptococci comprise a large and important group of organisms. They are Gram positive cocci which characteristically occur in chains, and frequently produce hemolysis on blood agar. Those producing beta hemolysis may be grouped by their polysaccharide antigen (C substance) into a number of groups entitled A, B, C and so on. Group B streptococci may be further subdivided into types, Ia, Ib, Ic, II and III.

These organisms were first found in cattle, especially in cases of mastitis, but it is now known that they form part of the normal flora of five to 15 per cent of human adults[1, 2]. They are most commonly found in the vagina, but also may be present in the throat, urethra or faeces of both males and females. Rarely they produce disease — post partum sepsis, osteomyelitis, meningitis — in their adult hosts.

PATHOGENESIS OF NEONATAL INFECTION

About two per cent of infants become colonized with Group B streptococci during delivery or shortly thereafter[2]. For the majority of infants these organisms become part of their normal flora and cause no harm. However, about one in ten of those colonized develop disease. Two clinical forms of disease are recognized: a fulminating septicemic form, which begins within 24 hours of birth, and a more insidious meningitic form which usually begins days or weeks after birth.

Infants who devclop the early, fulminating form of disease acquire their organisms before birth, from contaminated amniotic fluid, or during passage through the birth canal. The organisms are aspirated into the lung where they quickly multiply and enter the blood stream and spread throughout the body.

The pathogenesis is less clear in infants who develop Group B sepsis in the late neonatal period. Some acquire their organisms from their mother, but others apparently are infected from hospital personnel[2].

The pattern and severity of the disease appear to be type related. Group I subtypes seem responsible for most of the early onset, fulminating illnesses, while the late onset, meningitic forms seem related to type III organisms[2, 3].

CLINICAL MANIFESTATION

Predisposing maternal complications are frequently present in infants developing the early onset type of illness[3]. These include prolonged rupture of membranes and perinatal fever. Symptoms of respiratory distress and shock are usuallly present at birth or within the first 24 hours of life. Despite prompt treatment with appropriate antibiotics 70 to 100 per cent of infants die within two to three days[2, 4].

The late onset type of disease presents sometimes after the first week of life usually, in a more insidious manner. The meninges are almost always involved. Predisposing factors such as prematurity and maternal obstetrical complications seem less important in this disease. Lethargy, fever and bulging fontanelles are usual presenting signs. The prognosis is somewhat better in this type of infection, for the mortality is from 18 to 40 per cent[4, 5]. However, survivors may be left with serious neurologic sequellae, such as hydrocephalus, or mental retardation.

DISCUSSION

There is no apparent reason to explain why Group B streptococcus is causing an increased number of neonatal infections. It does not appear to be related to the use of antibiotics for these organisms are almost invariably sensitive to penicillin, the most widely used drug in the nursery. The number of adult asymptomatic carriers appears to be the same. One author[4] has suggested that the relative *amount* of organisms present in the vagina may have increased, possibly due to factors such as contraceptives, thus resulting in heavier colonization of the infant during delivery. It

*Head, Division of Microbiology, Women's College Hospital; Assistant Professor, Department of Medical Microbiology, Faculty of Medicine.

may be more than coincidence that the increasing infection rate with Streptococcus has coincided with the diminished use of hexachlorophene as a cleansing agent in the nursery.

A number of approaches have been put forward in an effort to control this disease. Some authors[2,5], noting that infants born of mothers harbouring the organism in their vagina are at risk of developing infection, have recommended that carrier mothers be identified by ante partum cultures and that they and their husbands be treated with penicillin. Others[4] have recommended culturing at birth infants of colonized mothers and treating those infants harbouring the organisms. Further prospective studies are needed to better define the epidemiology of Group B streptococcal infections in neonates in order to formulate effective treatment and control measures.

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For the Wellesley Hospital, it was a painful and unfortunate event, but we learned a great deal and were able to re-evaluate old procedures, correct faults and deficiencies and institute new and effective regulations.

The cause of the outbreak, however, still remains a mystery.

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